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THE PATHOLOGY AND TREATMENT OF SHOCK.

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THE PATHOLOGY AND TREATMENT OF SHOCK.*

BY EUGENE BOISE, M. D., GRAND RAPIDS, MICH.

"A specimen of ruptured heart was presented at the meeting of the Société Anatomique by Greffo. It was observed in the service of Chautemesse, by Menetrier. The patient, a woman of seventy-one years, was in apparent good health, but had a certain excess of adipose. It was suddenly announced to her that her son was very ill. She was immediately taken with choking, with syncope, and died. At the autopsy a certain quantity of blood was found effused into the pericardium, and a very neat rupture of the posterior wall of the left ventricle. The valves were healthy. The myocardium did not appear to be altered exteriorly." †

This is a case of sudden death from mental shock, and the interesting question immediately confronts us, What was the condition of the heart induced by the shock?

We are told that the pathology of shock is "paresis of the heart and abdominal vessels." ‡

Paresis of a muscle implies weakened contractile power. Rupture of a muscle by its own action is caused only by a sudden unusually violent contraction. Therefore, in the case above cited the shock must have caused sudden violent contraction of the heart; a condition resembling spasm rather than paresis.

This much by way of introduction to a further discussion of the pathology and treatment of shock.

What is the pathology of shock? is a question that has forced itself upon the attention of every observing surgeon. It is a question

* Read by invitation before the New York Obstetrical Society, January 7, 1896.

† *Medical and Surgical Reporter*, October 12, 1895.

‡ Gay. *Reference Handbook of the Medical Sciences.*

never definitely or completely answered, yet of prime importance to every operator. It has been almost universally accepted that it is some form of paresis of the nervous system.

In fact, Mr. Mansell-Moullin, in the *International Encyclopædia of Surgery*, says : "Shock is an example of reflex paralysis in the strictest and narrowest sense of the term, a reflex inhibition, probably in the majority of cases general, affecting all the functions of the nervous system, and not limited to the heart and vessels only."

Dr. George W. Gay says : * "Experimental physiology has demonstrated that in shock there is a reflex paralysis of the heart and abdominal vessels through the medium of the vasomotor system." Other writers give the same or similar pathology, but in no case is the reasoning entirely satisfactory, even apparently, to the authors themselves ; hence I have thought that further inquiry and argument might be of interest and benefit. Let me therefore introduce my discussion by this question : Is it not probable that the pathology of surgical shock is in no sense a *paresis*, but rather an *excessive irritation* of the entire sympathetic nervous system, manifested mainly by *excessive stimulation* of the vasomotor (constrictor) nerves ?

In the entire symptomatology of shock there are only two facts which apparently antagonize this theory—namely, the lowered blood pressure and the dulled cerebral and spinal functions, but that these are only apparently antagonistic I believe.

It seems to be an established physiological fact that all efferent non-medullated (sympathetic) nerves are excitor in their action ; that they tend to exert a steady tonic excitor action whether they be vasomotor or other. And, inasmuch as the great majority are known to have medullated fibers accompanying them, which pass through the ganglia unmodified, and whose action is inhibitory, it is a fair inference that every excitor sympathetic nerve has its corresponding inhibitory cerebro-spinal nerve, and it may be that inasmuch as there is one general vasomotor center, there may well be some general inhibitory center. Be that as it may, all inhibitory nerves (not speaking of the cranial nerves) arise from the anterior columns of the spinal cord, and pass through the corresponding sympathetic ganglia unchanged, to be lost finally in the terminal ganglionic cells, maintaining throughout their entire course the physical characteristics of spinal nerves.

But it is only by an analysis of the symptoms of shock in their re-

* *Reference Handbook of Medicine and Surgery.*

lation to the physiological action of the nervous system that we can arrive at the correct pathology. These symptoms are mainly—

1. Mental apathy. 2. Muscular weakness of the limbs, etc. 3. Lividity of skin, lips blue, finger nails blue, no waxy pallor. 4. Pupils *dilated.* 5. Perspiration. 6. Pulse almost imperceptible, unequal and very rapid; arteries small and tension low. 7. Decreased secretion of urine.

These are the main symptoms universally conceded to be characteristic of shock, and yet the article in the *International Encyclopædia of Surgery*, in which the accepted pathology of shock is very carefully discussed, says: "It may be taken as demonstrated beyond dispute that in shock there is a reflex paralysis of the heart and abdominal vessels," but admits that "there is more than this," "while the abdominal vessels are somewhat distended (sometimes) they are not enough so to account for the symptoms"; that "there is probably a paralysis of the entire vasomotor system," and concludes, as I have before quoted, that "shock is an example of reflex paralysis in the strictest and narrowest sense of the term, a *reflex inhibition*, probably in the majority of cases general, affecting all the functions of the nervous system, and not limited to the heart and vessels only."

I am obliged to admit that I can not perceive how an analysis of the symptoms, according to known physiological facts, will bear out this statement.

It would seem that the prevailing idea that shock is essentially a paresis is based upon—

1. The marked depression which undoubtedly exists in every well-marked case, especially of those portions of the system under the control of the brain and spinal cord directly.

2. The low arterial tension which exists. That there is mental apathy, muscular weakness (general), and dulled sensation is evident in every case, but it seems to me that this condition is not properly part of the pathology of shock, but rather a result or symptom merely, very like the reflex paraplegia described by Dr. Brown-Séquard. He says the paraplegic symptoms depend upon or are caused by a spinal anæmia due to tonic contraction of the arterioles supplying the part, and that this is in turn the direct result of *irritation* of the vasomotor nerves supplying those arteries.

If we accept this pathology of reflex paraplegia, as explained by so accurate an observer as Dr. Brown-Séquard, it follows as a logical conclusion that if local acute spinal anæmia causes paresis of the nerves below that point, acute general anæmia of the entire cerebro-

spinal system induced by general arterial contraction may cause general cerebro-spinal paresis.

That the entire arterial system is contracted all observers must admit, but that it can not be due to *hyperirritation* of the general vasomotor system they do not admit, because theoretically arterial tension should be high, whereas it is low. Moreover, the experiment of Goltz, of Strasbourg, seems to contradict it.

In this a frog, suspended legs downward, was struck sharply on the abdomen. The heart immediately stopped, and the abdominal vessels became intensely distended, owing to paralysis of their muscular coats.

Is it not possible that this experiment has been misinterpreted, and therefore has led to serious misapprehension? In the first place, whether the conditions (anatomical and physiological) in the frog and human being are the same I do not know, but I do know that in surgical shock, especially where induced by operations on the abdominal viscera, there is no marked increase in the distention of the abdominal vessels. This same observation has been made by others.

That a correspondingly severe blow on the epigastrium of a human being might result in conditions similar to those observed by Goltz I can conceive, but would it be surgical shock? Would it not rather be *concussion* of the solar plexus? A severe blow on the head suspends the activity of the nerve cells of the brain—concussion of the brain. A severe blow on the spine causes a more or less complete suspension of the functions of the nerve cells, etc., of the cord—concussion of the spine. The solar plexus is, like the brain and spinal cord, largely composed of nerve cells which, though they may not have the power of originating nerve force, most certainly have an important influence on its transmission and on the life of those fibers springing from them. Therefore a severe blow over the solar plexus might, and naturally would, cause suspension, more or less complete, of the power of its nerve cells, and consequently cause paralysis, more or less complete, of the nerves passing from them, with distention of the vessels supplied by those nerves. But that an *injury* to a nerve or plexus can transmit through afferent fibers an impulse of paresis which will be reorganized at the reflex center and transmitted as a paretic impulse to the entire sympathetic system, I can not understand. I think I am right in saying that any impulse originated by *traumatism*, which is capable of being reflected, is necessarily an impulse of stimulation.

The theory advanced many years ago by Dr. Weir-Mitchell and

others, that severe injury to nerves would cause an impulse of stimulation so excessive as to completely overwhelm the nerve centers, and thus cause reflex paralysis, may possibly serve to explain certain cases. I can conceive that the nerve cells from which the irritated fibers spring may be overwhelmed, but that this paretic condition can be so extensively reflected, or can involve so large an area of nerve centers as to cause the universal paretic condition, cerebral and spinal, found in all cases of severe shock, I can not believe.

But the heart was also markedly affected in Goltz' experiment. This would follow as a necessary physiological consequence of such a blow. The solar plexus receives fibers from the pneumogastric, the inhibitory nerve of the heart; some of these fibers are afferent, convey ing impressions to the medulla. A blow on the epigastrium would probably cause an impulse of stimulation to the afferent fibers passing through the solar plexus, and this impulse would certainly be reflected down the pneumogastric, causing inhibition of the heart. But all this is not surgical shock, as we see it as a result of abdominal operations or crushing injuries to the extremities.

Surgical shock is essentially due to a reflex impulse which is distributed not merely to the cardiac and vasomotor nerves, but to the entire sympathetic system, as I think an analysis of the symptoms will show.

The fact that arterial tension is undoubtedly low need not be a stumbling-block to us, nor should it bear weight in favor of vasomotor paresis in the face of the combined evidence of other conditions antagonistic to it, especially as it is capable of satisfactory physiological explanation.

Let me discuss the symptoms in the order of their occurrence as nearly as may be. First occurs the injury—a crushing injury to the limbs, it may be—or a laceration or contusion of sympathetic fibers in the abdomen. Not only are the nerves beyond the point of injury strongly irritated, but an impulse of intense irritation passes along the afferent nerves to the corresponding nerve centers, and is so severe that the entire reflex centers of the cord, from which the efferent sympathetic fibers arise, are intensely stimulated, and an impulse of extreme stimulation is transmitted to the entire vasomotor and sympathetic system.

As a consequence, the arterioles of the entire body are thrown into a condition of extreme constriction, causing a thready, almost imperceptible pulse. If the impulse were of paresis, the arteries would of necessity be dilated. No fact in physiology is better known than that

paralysis of vasomotors causes dilatation of corresponding arteries. At the same time the augmentor nerves of the heart are intensely stimulated, and, as a necessary physiological consequence, the heart's action becomes very rapid and its contractions almost tetanic. It contracts strongly and relaxes imperfectly—an exaggerated systole and incomplete diastole. By reason of its imperfect relaxation it receives a scanty supply of blood and sends into the arteries at each contraction only a small percentage of the normal quantity of blood As a consequence, the arteries are not at all distended and the blood pressure is necessarily *low*. This is not an imaginary explanation nor merely plausible, but is more than probable, as the same condition of the heart has been experimentally caused by electrical stimulation of its excitor nerves.*

Now, having these contracted arterioles, with limited supply of blood, what conditions would necessarily follow? First, acute anæmia of the brain and spinal cord, causing partial suspension of their functions (a condition which is marked in severe cases of shock), slowness of intellectual faculties, great muscular weakness, and partial anæsthesia.

This condition, then, is not part of the pathology of shock, but rather a symptom which will pass away when the cause is removed, exactly as in the cases of reflex paraplegia so ably discussed by Dr. Brown-Séquard.

Again, the decreased or even suspended secretion of urine is a direct result of contracted renal arterioles.

It has never been demonstrated that there are any nerves which have any direct influence on the secretory function of the kidney other than its vasomotor nerves. The amount of urine secreted, therefore, depends entirely on the vascular conditions; dilatation of the renal arterioles or increased velocity of the current, causing increased secretion of urine; contracted arterioles, especially if connected with low pressure, causing decreased secretion.

Inhibition or paralysis of the abdominal vessels would cause increased blood supply to the kidney and increased secretion of urine, especially if the arteries of the rest of the system were contracted.

Again, in surgical shock we have a livid pallor (if I may so express it) of the skin, with blueness of the lips and finger nails, and coldness of the surface. This is also a direct and necessary result of general arterial contraction. The smaller arteries have the most marked

* Landois and Sterling. *Manual of Physiology*, p. 106.

muscular coat, and are therefore most severely contracted under vasomotor stimulation. This, with imperfect relaxation of the heart, throws the blood into the veins and capillaries, which become generally distended, or at least disproportionately filled.

All these symptoms, which are present in every severe case of shock, are natural and necessary consequences of general contraction of the arterioles, or general vasomotor irritation, and could not occur if the condition was one of vasomotor paresis. One of the best known of vasomotor experiments is, that section of the cervical sympathetic, with consequent paralysis of the vasomotor nerves beyond that point, causes intense arterial hyperæmia of the tissues supplied by those nerves.

But we have other symptoms not dependent on vasomotor influences, but which just as evidently are caused by excessive stimulation of the sympathetic system. In shock the pupils are *dilated.* Section of the cervical sympathetic causes paralysis of the nerves beyond the point of section and *contraction* of the pupil. Stimulation of the cervical sympathetic always causes dilatation of the pupil.

Again, in shock there is free prespiration. Special secretory nerves, non-medullated, branches of the sympathetic system, have been demonstrated as distributed to the sweat glands, stimulation of which will cause increased perspiration without reference to the vascular conditions.

If in shock there is hyperirritation of the entire sympathetic system, these secretory fibers would be irritated, and there would be increased perspiration, a condition almost invariably present.

Again, there is often relaxation of the sphincters in severe shock. This condition implies a mixed nervous action. Normally, the sphincters are largely under the control of the will, and even though there be increased peristaltic action above them, the sphincters will not be relaxed. On the other hand, even if voluntary control of the sphincters be lost, as by cerebral concussion, there will not necessarily be any evidence of their relaxation, because peristalsis may be weakened or quiescent. But in excessive stimulation of the sympathetic system there is increased activity of the muscles of the intestines, which are directly under the control of this system (as has been conclusively proved), increased peristalsis. If, in addition to this, there is acute cerebral anæmia, with consequent impairment of volition, the voluntary control of the sphincters would be suspended, and, with increased peristalsis, there might well be involuntary evacuations.

Thus a careful, unprejudiced analysis of the prominent symptoms

of shock, based on what we know to be universally accepted anatomical and physiological facts, forces me to the conclusion that there is no other correct interpretation of the phenomena of shock than that based on the theory of hyperirritation of the entire sympathetic system.

If it be true that there is a general inhibitory system, the primary factor might be general *paralysis of inhibition*, with consequent *secondary* hyperstimulation or augmentation of the entire non-medullated system, if it were not for the fact that (according to Foster) experiments tend to show that, while there is a constant tonic activity of the *vaso-constrictor* fibers, there does not appear to be any such constant action of the *vaso-dilators* (inhibitory).

I therefore can not but conclude that the *primary* and essential factor in surgical shock is hyperirritation of the entire sympathetic system.

A consideration of the treatment of shock will bring us to the same conclusion.

Clinical experience has taught us that the remedies most useful in its treatment are, of drugs, nitrite of amyl, nitroglycerin, opium, and strychnine. Of other agents, the external application of heat, the rectal injection of water as hot as can be borne, and intravenous transfusion of saline solution at the temperature of 118° or 120°.

It is beyond question that nitrite of amyl acts as a rapid but evanescent relaxant to the muscular coat of the arterioles. The flushed face and sense of cerebral "fullness" which follows a few inhalations is satisfactory evidence of this action. When, during a severe abdominal operation, shock supervenes, as shown by the rapid, thready pulse, nothing brings such speedy relief as inhalation of nitrite of amyl, but the evanescent nature of the remedy precludes any dependence on it for a prolonged effect. To those who have had occasion to use the remedy in these conditions no description of its effect is necessary. Clinical experience speaks loudly for it as a rapid dilator of contracted arterioles.

Nitroglycerin, another member of the same group, has long been known as a "heart tonic," not by reason of its special action on the heart, but because of its relaxing effect on the arterioles, thus relieving an overburdened heart. There has never been any question as to its paralyzing action on the muscular coats of the arterioles, and it is universally conceded that it is one of the best remedies in surgical shock.

If it be true that the pathology of shock is a hyperirritation of

the entire sympathetic system, we should expect contracted arterioles by reason of tonic spasm of their muscular coats; and we should also expect benefit from nitrite of amyl and nitroglycerin because of their undisputed action as relaxants of arterial spasm. The reputation of opium, while firmly established in the estimation of many surgeons as an invaluable remedy in the treatment of shock, is not so unquestioned as an arterial relaxant. And yet physiological researches have fully proved that in full doses it exerts a marked sedative action on the vasomotor system, and not only on the vasomotor, but on the entire sympathetic system.

Therefore, if the pathology of surgical shock be hyperirritation of the entire sympathetic system, it can be easily understood how opium may be an invaluable agent for its relief.

Strychnine has long been held in high esteem as a remedy of great value in the treatment of shock, many operators placing their main reliance on it. A consideration of its action, as demonstrated by physiological experiment, shows that its use is not only not incompatible with the irritant theory of shock, but is strongly confirmative of it.

Van Deen, by his experiments on animals, has shown that strychnine stimulates the motor centers of the spinal cord. Other experiments have confirmed this, so that it is undeniable that the main feature of its action is stimulation to the cerebro-spinal system of nerves, especially to the anterior columns of the spinal cord.

In toxic doses the effect is almost entirely confined (apparently) to this cerebro-spinal system. In the anatomy of the vasomotor system the nerves which act as vaso-constrictors are invariably non-medullated and arise from the cells of the sympathetic ganglia. In other words, they are purely fibers of the sympathetic nervous system. Excitation of the entire sympathetic system would therefore cause excitation of these fibers, and contraction of the muscular coats of the arteries to which they are supplied.

Accompanying each *vaso-constrictor* sympathetic nerve fiber there is always another nerve fiber which arises from the *anterior columns* of the spinal cord and passes through the sympathetic ganglia without becoming connected with any of its nerve cells, and reaches its destination in the cells of the ganglia of the muscular coats of the arteries. It is medullated throughout its entire course, and is anatomically, in all respects, a spinal nerve. It is always *inhibitory* in its action, or vaso-dilator.

Therefore, if shock is characterized by spasm of the muscular coats

of the arteries, due to excitation of the vaso-constrictor sympathetic fibers, strychnine, by reason of its peculiar affinity for the cerebro-spinal system, especially the anterior columns of the spinal cord, would disproportionately stimulate these spinal *inhibitory* fibers, paralyzing, as it were, the ganglia of the arterial muscles, and tend to antagonize or overcome arterial spasm, and in this way would be a valuable agent in the treatment of shock.

"It has been shown by the experiments of Klapp that the primary stimulation of the vasomotor centers (by moderate doses of strychnine) is followed by fall of arterial pressure and vasomotor palsy. Also that very large doses produce an immediate depression of the vasomotor centers and fall of the arterial pressure."*

His experiments also show that not only do large doses paralyze the vasomotor center, but they slow the pulse by an immediate paralyzing action on the excitor ganglia of the heart.

Dr. Carl Heineman has also found that "large doses cause diminished frequency of the cardiac movements with diastolic pauses."

La Housse also, by his experiments on dogs, has demonstrated that sulphate of strychnia slows the action of the heart by reason of a paralyzing influence on the intracardiac ganglia—exactly the same influence that I have endeavored to show that it exerts on the ganglia of the muscular coats of the arteries. Now, according to the irritant theory of the nature of shock, there is not only arterial spasm, but there is also cardiac spasm—the heart contracting strongly and relaxing imperfectly—exaggerated systole, and incomplete diastole. If this theory be the true one, and if the deductions of La Housse and others be correct, we should expect strychnine in the large doses always used to be of great value, not only because of its stimulating action to the vaso-dilators (or paralyzing action on the terminal vasomotor ganglia), but by reason of its paralyzing action on the intracardiac ganglia, thus relieving cardiac spasm and allowing full and normal relaxation.

Strychnine, therefore, falls naturally into the group of arterial and cardiac relaxants, and justifies its selection as one of our most valuable remedies in the treatment of shock.

An analysis of the action of the other remedies mentioned (external heat and rectal and intravenous injections of hot water) will not only bear out this hyperirritation theory of shock, but almost necessitates its recognition.

* H. C. Wood. *Therapeutics and Pharmacology.*

All operators admit the usefulness of heat to the surface, but say that it is used to "stimulate the circulation."

This term is vague, and in this connection means nothing. The desire is to restore warmth to the skin and bring about a condition of arterial hyperæmia. To do this, external heat must be judiciously applied. It must not be so intense as to overstimulate the ganglia in these cutaneous arterioles and cause further contraction, but must be of such degree and nature as to "invite" the blood to the surface.

Section of the cervical sympathetic causes paralysis of the vasomotor nerves beyond that point, and consequent arterial hyperæmia (intense flushing) of the corresponding side of the head. Heat applied to the surface of the body accomplishes the same purpose by sedation of the ganglia of the cutaneous arterioles, thus allowing their relaxation, which is manifested by arterial hyperæmia of the skin.

Heat applied to the surface of the body, therefore, acts as a local sedative to the cutaneous arterioles and tends to their relaxation. Whether this influence of sedation is transmitted through afferent nerves and reflected to other portions of the vasomotor system I do not know.

The action of rectal injections of water as hot as the patient can bear, and the intravenous injections of saline solution at 118° or 120°, as recommended by Dr. Dawbarn, are virtually the same, and are among the most valuable agents in the treatment of surgical shock. The action is twofold: First, there is an influence on the vasomotor nerves, and, second, fluid is supplied to the blood vessels.

In nearly all cases of surgical shock, whether consequent upon abdominal operations or due to severe crushing injuries, there is a depleted condition of the circulation—in the one case due to the anæmia consequent on a long and exhausting illness, it may be, or in the other caused by hæmorrhage consequent on the injury. In both cases it is advisable to supply fluid to the system. In urgent or aggravated cases, where the danger is imminent, intravenous transfusion of hot saline solution is a remedy of inestimable value. The action, as I have said, is twofold: First, supplying the depleted vessels with fluid, and, second, the action of the heat on the vasomotor nerves. The first indication is met equally well by both methods, more rapidly by transfusion, but equally well, though more slowly, by rectal injection.

The second indication—namely, the action of heat on the vasomotor nerves—is met by the two methods in somewhat different ways and degrees.

Dr. Dawbarn, in advocating intravenous transfusion, stated that the saline solution should be about 118° or 120° F., as at that temperature the solution would act as a *stimulant* to the cardiac and arterial ganglia. Practically he was correct, but theoretically he was wrong.

Diluted by admixture with the blood, the temperature would be materially reduced, and a *moderate* degree of heat would be *sedative*. Fortunately, this is what we want. The hot saline solution, mingled with the blood, is brought into intimate relation with the ganglia of the cardiac and arterial muscles, and by its sedative influence on these ganglia promotes relaxation of the spasmodically contracted muscular fibers, allowing an equalization of the blood supply and relieving shock.

If the water is introduced into the system *per rectum* it should be as warm as can be borne, and introduced through the long tube. In this way not only is water supplied to the depleted vessels, but the heat is brought up to the solar plexus, or near it, and exercises a sedative influence on that important aggregation of ganglionic cells and fibers.

Thus all remedies of greatest repute in the treatment of surgical shock bring the weight of their clinical evidence to the support of the theory that surgical shock is essentially a hyperirritation of the entire sympathetic nervous system.

Let me give a hasty *résumé* of the manner in which I think surgical shock should be treated.

First, the inhalation of nitrite of amyl not only while the patient is on the operating table, but repeated at intervals until the full effect of other remedies is obtained.

Second, the hypodermic injection of nitroglycerin in doses that ordinarily would be almost toxic.

I think we often fail by not appreciating the severity of the arterial spasm that is to be overcome.

With this, if the case is not urgent, rectal injections of hot saline solution repeated as often as the bowel will tolerate it, and administered in such manner that the fluid will rapidly pass into the transverse colon. If the case be urgent, the fluid must be thrown into a vein. In my opinion, no remedy is of more value than hot saline solution thus administered.

Finally, sulphate of strychnia administered hypodermically in doses regulated by the indications of each case.

All drugs should be administered hypodermically.

In all cases about to undergo a severe operation I anticipate the occurrence of shock, and in a measure attempt to prevent it by the hypodermic administration just before the anæsthetic of one or one and a half grains of codeine, and am convinced that it has a very favorable effect.

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